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Issue Date: 20 July 2006

In the Matter of:

PEGGY J. HOWERTON, widow of
ROOSEVELT HOWERTON
Claimant

Case No.: 2005 BLA 23

v.

EASTERN ASSOCIATED COAL COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party in Interest

Appearances: Mr. John Cline, Attorney
For the Claimant

Mr. Paul E. Frampton, Attorney
For the Employer

Before: Richard T. Stansell-Gamm
Administrative Law Judge

DECISION AND ORDER –
DENIAL OF MODICATION REQUEST

This matter involves a claim filed by Mrs. Peggy Howerton, widow of Mr. Roosevelt Howerton, for survivor benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 ("the Act"). Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as "black lung" disease.

Procedural Background

Mr. Howerton's Black Lung Disability Claims

Initial Claim
(DX 1)[1]

On July 3, 1980, Mr. Howerton filed his first claim for black lung disability benefits. Following a pulmonary examination, on May 1, 1981, the U.S. Department of Labor ("DOL") determined Mr. Howerton qualified for black lung disability benefits. However, because Mr. Howerton remained employed as a coal miner, an award of benefits could not be made unless he agreed to stop mining coal within one year. On May 7, 1981, the Employer agreed to accept responsibility for payment of benefits once Mr. Howerton stopped coal mining. On July 16, 1982, DOL denied Mr. Howerton's claim because he was not totally disabled due to coal workers' pneumoconiosis since he failed to stop mining coal within one year of the initial determination of his qualification for benefits.

Second Claim
(DX 2)

On August 11, 1986, Mr. Howerton filed a second claim. On January 15, 1987, DOL determined Mr. Howerton was entitled to black lung disability benefits. On February 2, 1987, the Employer accepted responsibility for payment and DOL issued a final award determination on February 18, 1987. The Employer continued payments until May 1, 1999, the month Mr. Howerton passed away.

Mrs. Howerton's Survivor Claim

After her husband's death on May 27, 1999, Mrs. Howerton filed a survivor claim for benefits on June 14, 1999 (DX 3). On November 30, 1999, her claim was denied because the medical evidence failed to establish that her husband's death was due to coal workers' pneumoconiosis (DX 18). On December 18, 1999, an attorney[2] for Mrs. Howerton requested a hearing with the Office of Administrative Law Judges ("OALJ") (DX 18). After several additional requests for a hearing, the case was forwarded to OALJ on May 1, 2002 (DX 18,) On October 9, 2002, Administrative Law Judge Michael Lesniak conducted a hearing. On April 2, 2003, Judge Lesinak denied Mrs. Howerton's survivor claim because the medical evidence was insufficient to establish that her husband's death was due to coal workers' pneumoconiosis (DX 33). On April 14, 2003, Mrs. Howerton appealed the adverse decision (DX 34). On January 30, 2004, the Benefits Review Board ("BRB" and "Board") affirmed Judge Lesniak's denial of survivor benefits (DX 40).

[1]The following notations appear in this decision to identify exhibits: DX – Director exhibit; CX – Claimant exhibit; EX – Employer exhibit; TR – Transcript; and, ALJ – Administrative Law Judge exhibit.

[2]At that time, Mrs. Howerton was represented by Mr. S. F. Raymond Smith.

On May 4, 2004, Mrs. Howerton submitted a modification request (DX 42). On September 14, 2004, DOL denied the modification request, finding the preponderance of medical opinion indicated that Mr. Howerton's death was not due to coal workers' pneumoconiosis (DX 47). Mrs. Howerton appealed the adverse decision and the case was returned to OALJ on December 13, 2004 (DX 48 and DX 51). Pursuant to a revised Notice of Hearing, dated April 12, 2005, I conducted a hearing on May 10, 2005 in Princeton, West Virginia, with Mrs. Howerton, Mr. Cline, and Mr. Frampton present. My decision in this case is based on the hearing testimony and the following documentary evidence: DX 1 to DX 52, CX 1 to CX 6, and EX 1 to EX 4.[3]

Procedural Comment

During the initial adjudication of Mrs. Howerton's survivor claim, Judge Lesniak discussed the issue of collateral estoppel in some detail. Specifically, he considered whether in the absence of an autopsy and due to the award of black lung disability benefits to Mr. Howerton, the Employer was precluded from relitigating whether Mr. Howerton had pneumoconiosis. Based on his review of the case law, Judge Lesniak indicated collateral estoppel may be warranted. However, because the medical evidence on the cause of Mr. Howerton's death was insufficient to establish death due to pneumoconiosis, Judge Lesinak believed the collateral estoppel issue was not pivotal to the resolution of the survivor claim and did not actually make a final decision on collateral estoppel. Similarly, during consideration of Mrs. Howerton's appeal, the Benefits Review Board did not discuss the issue of collateral estoppel.

Based on more recent case law, I conclude collateral estoppel does not apply in Mrs. Howerton's survivor claim. Since Mr. Howerton last mined coal in West Virginia, Mrs. Howerton's survivor claim falls under the jurisdiction of the U.S. Court of Appeals for the Fourth Circuit.[4] In *Howard v. Valley Camp Coal Co.*, Case No. 03-1706 (4th Cir. Apr. 14, 2004) (unpub.), that court concluded an employer was not collaterally estopped from re-litigating the presence of pneumoconiosis provided the award of black disability benefits to the deceased miner pre-dated the issuance of *Island Creek Coal Co. v. Compton,* 211 F.3d 203 (4th Cir. 2003).[5] Accordingly, since Mr. Howerton was awarded black lung disability benefits in 1987, and in the absence of any stipulation concerning the presence of pneumoconiosis, the Employer is not estopped from contesting the presence of pneumoconiosis in Mr. Howerton's lungs during the present adjudication of Mrs. Howerton's survivor claim.

[3]As approved, post-hearing, I received Dr. Branscomb's June 1, 2005 deposition which I admit as EX 4.

[4]*See Shupe v. Director, OWCP,* 12 B.L.R. 1-200, 1-202 (1989) (en banc)

[5]Rather permitting the presence of pneumoconiosis to be determined by any one of four methods under 20 C.F.R. § 781.202 (a) (1)-(4), the *Compton* decision requires a fact finder to weigh evidence under all four methods in determining whether pneumoconiosis is present.

ISSUE

Whether in filing a modification request on May 4, 2004, Mrs. Howerton has demonstrated a mistake in determination of fact occurred in the affirmed denial of her survivor claim by Administrative Law Judge Michael Lesniak on April 2, 2003.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Stipulations of Fact

At the May 10, 2005 hearing, the parties stipulated: a) Mr. Howerton engaged in post-1969 coal mine employment; b) Mr. Howerton had at least 24 years of coal mine employment; c) Mrs. Peggy Howerton is an eligible survivor under the Act; and, d) Eastern Associated Coal Company is the responsible operator (TR, pages 8, 9, and 23).

Preliminary Findings

Born on January 24, 1935, Mr. Howerton married Mrs. Peggy Howerton on June 2, 1956. He started mining coal in 1953. After about a year, Mr. Howerton entered the military and did not return to coal mining until 1956. After working for one year as a coal miner, Mr. Howerton left the mines from 1957 to 1962. Mr. Howerton returned to coal mining in 1962 and continued through January 28, 1986, when the coal mine shut down. During his coal mining career, Mr. Howerton worked as a roof bolter, beltman, and boom man. He smoked cigarettes between 20 to 35 years at the rate of one half to two packs per day. Mr. Howerton passed away on May 27, 1999 (DX 1, DX 2, DX 3, DX 10, and TR, pages 17 to 22).

Issue #1 – Modification

Any party to a proceeding may request modification at any time before one year from the date of the last payment of benefits or at any time before one year after the denial of a claim. 20 C.F.R. § 725.310 (a) (pre-2001).[6] Upon the showing of a "change in conditions" or a "mistake in a determination of fact," the terms of an award or the decision to deny benefits may be reconsidered. 20 C.F.R. § 725.310 (pre-2001). An order issued at the conclusion of a modification proceeding may terminate, continue, reinstate, increase or decrease benefit payments or award benefits.

Since the present modification request relates to Mrs. Howerton's survivor claim, evaluation of the record for a change in conditions is not warranted.[7] Instead, the focus in modification proceedings in a survivor claim concerns a mistake of fact analysis. In *O'Keefe v.*

[6]In January 2001, a new set of DOL regulations concerning the adjudication of black lung claims became effective. Most of the new regulations in 20 C.F.R. Part 718 and some portions of Part 725 are applicable to Mrs. Howerton's pending survivor claim. However, the old version of 20 C.F.R. § 725.310, which I have designated with the suffix "(pre-2001)," controls the procedural status of Mr. Howerton's survivor claim. *See* 20 C.F.R. § 725.2 (c).

[7]Because Mr. Howerton has passed away in 1999, there can be no change in conditions concerning his pulmonary condition since the denial of Mrs. Howerton's survivor claim in 2003.

Aerojet-General Shipyards, Inc., 404 U.S. 254, 257 (1971), the United States Supreme Court indicated that an administrative law judge should review all evidence of record to determine if the original decision contained a mistake in a determination of fact. In considering a motion for modification, the administrative law judge is vested "with broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." See also *Jessee v. Director, OWCP*, 5 F.3d 723 (4th Cir. 1993); *Director, OWCP v. Drummond Coal Co. (Cornelius)*, 831 F.2d 240 (11th Cir. 1987).

My determination of whether a mistake in determination of fact occurred during the prior adjudication of Mrs. Howerton's survivor claim involves the four entitlement elements that a claimant must prove by a preponderance of the evidence to receive survivor benefits under the Act and 20 C.F.R. § 718.205 (a). The claimant bears the burden of establishing these elements by a preponderance of the evidence. If the claimant fails to prove any one of the requisite elements, the survivor claim for benefits must be denied. *Gee v. W. G. Moore and Sons*, 9 B.L.R. 1-4 (1986) and *Roberts v. Bethlehem Mines Corp*., 8 B.L.R. 1-211 (1985).

First, the claimant must establish eligibility as a survivor. A surviving spouse may be considered eligible for benefits under the Act if she was married to, and living with, the coal miner at the time of his death, and has not remarried.[8]

Second, the claimant must prove the coal miner had pneumoconiosis.[9] "Pneumoconiosis" is defined as a chronic dust disease arising out of coal mine employment. The regulatory definitions include both clinical pneumoconiosis (the diseases recognized by the medical community as pneumoconiosis) and legal pneumoconiosis (defined by regulation as any chronic lung disease arising out of coal mine employment).[10] The regulation further indicates that a lung disease arising out of coal mine employment includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."[11] As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

Third, once a determination has been made that a miner had pneumoconiosis, it must be determined whether the coal miner's pneumoconiosis arose, at least in part, out of coal mine employment.[12]

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[8]20 C.F.R. § 718.4 indicates that the definitions in 20 C.F.R. § 725.101 are applicable. 20 C.F.R. § 725.101, in turn, refers to the term "survivor" as used in Subpart B of Part 725. 20 C.F.R. § 725.214 then sets out the spousal relationship requirements and 20 C.F.R. § 725.215 describes the dependency rules. According to § 725.214 (a) the spousal relationship exists if the relationship is a valid marriage under state law. Under § 725.215(a), a spouse is deemed dependent if she was residing with the miner at the time of his death.

[9]20 C.F.R. § 718.205 (a) (1) and see *Trumbo v. Reading Anthracite Co*., 17 B.L.R. 1-85 (1993).

[10]20 C.F.R. § 718.201 (a) (1) and (2).

[11]20 C.F.R. § 718.201 (b).

[12]20 C.F.R. §§ 718.203 (a) and 205 (a) (2).

Fourth, the surviving spouse has to demonstrate the coal miner's death was due to pneumoconiosis.[13]

Eligible Survivor

Based on the parties' stipulation, and her hearing testimony, I find Mrs. Howerton is an eligible survivor under the Act.

Pneumoconiosis

As a preliminary comment, in light of the present medical consensus that Mr. Howerton's pulmonary impairment was at least a contributing cause of death, the pivotal issue in this case is whether that pulmonary impairment involved either coal workers' pneumoconiosis or his inhalation of coal mine dust during the course of his career as a coal miner.

According to 20 C.F.R. §718.202, the existence of pneumoconiosis may be established by four methods: chest x-rays (§ 718.202 (a)(1)), autopsy or biopsy report (§ 718.202 (a)(2)), regulatory presumption (§ 718.202 (a)(3)),[14] and medical opinion (§ 718.202 (a)(4)). Mrs. Howerton filed her survivor claim after January 1982 and no autopsy was conducted in this case. As a result, Mrs. Howerton will have to rely on the regulatory presumption associated with complicated pneumoconiosis, chest x-rays, or medical opinion to establish the presence of pneumoconiosis. Additionally, under the guidance of *Compton*,[15] I must consider all the medical evidence together to determine whether the presence of pneumoconiosis in Mr. Howerton's lungs has been established.

Regulatory Presumption – Complicated Pneumoconiosis

The regulation, in part, at 20 C.F.R. § 718.304, provides that if a claimant is able to establish the presence of complicated pneumoconiosis, then an irrebuttable presumption of total disability and death due to pneumoconiosis is established. In the Black Lung Benefits Act, 30 U.S.C. 921 (c) (3) (A) and (C), as implemented by 20 C.F.R. § 718.304 (a), Congress determined that if a miner suffered from a chronic dust disease of the lung which "when diagnosed by chest X-ray, yields one or more large opacities (greater than one centimeter in diameter) and would be classified in category A, B, or C," there shall be an irrebuttable presumption that his death was due to pneumoconiosis.[16] This type of large opacity is called "complicated pneumoconiosis."

[13]20 C.F.R. § 718.205 (a) (3).

[14]If any of the following presumptions are applicable, then under 20 C.F.R. § 718.202 (a)(3), a miner is presumed to have suffered from pneumoconiosis: 20 C.F.R. § 718.304 (if complicated pneumoconiosis is present, then there is an irrebuttable presumption that the miner is totally disabled due to pneumoconiosis); 20 C.F.R. § 718.305 (for claims filed before January 1, 1982, if the miner has fifteen years or more coal mine employment, there is a rebuttable presumption that total disability is due to pneumoconiosis); and 20 C.F.R. § 718.306 (a presumption when a survivor files a claim prior to June 30, 1982).

[15]*See Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000).

[16]On the standard ILO chest x-ray classification worksheet, Form CM 933, large opacities are characterized by three sizes, identified by letters. Category A indicates the presence of a large opacity having a diameter greater than 10

The statute and regulation, 30 U.S.C. 921 (c) (3) (B) and (C) and 20 C.F.R. §§ 718.304 (b) and (c), also permit complicated pneumoconiosis to be established by either the presence of massive fibrosis in biopsy and autopsy evidence or other means which would be expected to produce equivalent results in chest x-rays or biopsy/autopsy evidence.

According to the U.S. Court of Appeals for the Fourth Circuit in *Eastern Associated Coal Corp. v. Director, OWCP [Scarbro]*, 220 F.3d 250 (4th Cir. 2000), the existence of complicated pneumoconiosis is established by "congressionally defined criteria." As a result, the statute's definition of complicated pneumoconiosis as radiographic evidence of one or more large opacities categorized as size A, B, or C, 30 U.S.C. 921 (c) (3) (A), represents the most objective measure of the condition. This sets the benchmark by which other methods for proving complicated pneumoconiosis are measured, as described in 30 U.S.C. 921 (c) (3) (B) and (C). *Id*. at 256. In other words, whether a massive lesion or other diagnostic results represent complicated pneumoconiosis under 30 U.S.C. 921 (c) (3) (B) and (C) requires an equivalency evaluation with the x-ray criteria set forth in 30 U.S.C. 921 (c) (3) (A).[17] Additionally, the court emphasized that the legal definition of complicated pneumoconiosis as established by Congress controls over the medical community's definition of the disease. *Id*. at 257. Finally, the court indicated that although all relevant and conflicting medical evidence must be considered and evaluated:

> if the x-ray evidence vividly displays opacities exceeding one centimeter, its probative force is not reduced because the evidence under some other prong is inconclusive or less vivid. Instead, the x-ray evidence can lose force only if other evidence affirmatively shows that the opacities are not there or are not what they seem to be, perhaps because of an intervening pathology, some technical problem with equipment, or incompetence. *Id*.

Referencing a 1993 case from the U.S. Court of Appeals for the Fourth Circuit, *Lester v. Director, OWCP,* 993 F.2d 1143, 1145-46 (4th Cir. 1993) the Benefit Review Board recently emphasized in *Mullins v. Plowboy Coal Company,* No. 04-0716 BLA, (unpub.), July 8, 2005, that an administrative law judge "must weigh together all of the evidence relevant to the presence or absence of pneumoconiosis." That mandate is consistent with other case law indicating that all evidence relevant to whether the miner has pneumoconiosis must be weighed. *Gray v. SLC Coal Co*., 176 F.3d 382 (6th Cir. 1999), *Melnick v. Consolidation Coal Co.,* 16 B.L.R. 1-31 (1991); *Maypray v. Island Creek Coal Co.*, 7 B.L.R. 1-683 (1985).

In other words, even if the presence of large opacities has been established through one of the three methods set out in § 718.304, all other medical evidence must be considered and evaluated to determine whether the large opacities actually exist and involve pneumoconiosis. For example, the Benefits Review Board affirmed a finding of complicated pneumoconiosis

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mm (one centimeter) but not more than 50 mm; or several large opacities, each greater than 10 mm but the diameter of the aggregate does not exceed 50 mm. Category B means an opacity, or opacities "larger or more numerous than Category A" whose combined area does not exceed the equivalent of the right upper zone of the lung. Category C represents one or more large opacities whose combined area exceeds the equivalent of the right upper zone.

[17]See also 20 C.F.R. §§ 718.304 (b) and (c).

under 20 C.F.R. §718.304 when the administrative law judge considered chest x-rays in conjunction with CT-scan findings to determine there was sufficient evidence to find complicated pneumoconiosis. *Keene v. G&A Coal Co.*, BRB No. 96-1689 BLA (Sept. 27, 1996). And, in another case, despite radiographic evidence of large opacities, the U.S. Court of Appeals for the Sixth Circuit upheld a determination that complicated pneumoconiosis did not exist based on probative autopsy evidence indicating the lesions were not complicated pneumoconiosis. *Gray*, 176 F.3d at 388.

In light of these statutory, regulatory and judicial principles, my present adjudication of whether a claimant is able to invoke the irrebuttable presumption under 20 C.F.R. § 718.304 involves a three step process. First, I must determine whether: a) the preponderance of the chest x-rays establishes the presence of large opacities characterized by size as Category A, B, or C under recognized standards; or b) biopsy evidence shows massive fibrosis; or c) other diagnostic results exist which are equivalent to the requisite chest x-ray or biopsy evidence of large opacities.

Second, if radiographic, biopsy or other equivalent evidence of large opacities exists, I must evaluate all the other relevant evidence in the record to determine whether it confirms or contradicts the presence of large opacities. In other words, I must assess whether the preponderance of the entire evidentiary record establishes the presence of large pulmonary opacities.

Third, if the preponderance of the evidence does demonstrate the existence of large opacities, I must then consider all other relevant evidence to determine whether that evidence contradicts or supports a finding that the large opacities are indicative of complicated pneumoconiosis.

Existence of Large Opacities

In the absence of biopsy/autopsy evidence or other supportive medical tests,[18] Mrs. Howerton must rely on chest x-ray imaging to establish the presence of large opacities.

Date of x-ray	**Exhibit**	**Physician**	**Interpretation**
April 11, 1978	DX 1	(WV Occupational Pneumoconiosis Board)	(Positive for pneumoconiosis). Moderately extensive reticular nodular fibrosis, consistent with occupational pneumoconiosis. (No large opacities reported.)
August 11, 1979	DX 1	Dr. Merchant	(Positive for pneumoconiosis) Category 1, simple pneumoconiosis present. (No large opacities reported.)
June 11, 1980	DX 1 & DX 12	Dr. Bassali, BCR,. B[19]	Positive for pneumoconiosis, profusion 2/2,[20] type p opacities.[21] (No large opacities reported).

[18]Dr. Rana reported complicated pneumoconiosis as part of Mr. Howerton's medical history upon his hospitalization admission in April 1997. However, since Dr. Rana did not indicate the underlying medical evidence or tests that supported the medical history, his entry does not establish the presence of complicated pneumoconiosis.

[19]The following designations apply: B – B reader, and BCR – Board Certified Radiologist. These designations indicate qualifications a person may posses to interpret x-ray film. A "B Reader" has demonstrated proficiency in

December 16, 1981	DX 2	(WV Occupational Pneumoconiosis Board)	(Positive for pneumoconiosis) Extensive irregular nodular fibrosis, consistent with occupational pneumoconiosis. (No large opacities reported.)
August 27, 1986	DX 2	Dr. Bassali, BCR, B	Positive for pneumoconiosis, profusion category 3/3, type q/r opacities. Large opacities, size A, both upper lung fields.
(same)	DX 2	Dr. Gaziano, B	Positive for pneumoconiosis, profusion category 2/3, type s/t opacities. No large opacities present.
(same)	DX 2	Dr. Sargent, BCR, B	Positive for pneumoconiosis, profusion category 1/1, type r/t opacities. No large opacities present.
April 3, 1997	DX 16	Dr. Fowler	COPD (chronic obstructive pulmonary disease), interstitial scarring, dense nodular infiltrate present. (No large opacities reported).
(same)	DX 16	Dr. Aycoth	Chronic interstitial fibrotic changes, large 5 cm mass right upper lung.
(same)	DX28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
April 4, 1997	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
April 6, 1997	DX 16	Dr. Aycoth	Chronic interstitial fibrotic changes, large 5 cm mass right upper lung.
(same)	DX 16	Dr. Ahmed	(Negative for pneumoconiosis). Advanced COPD, dense opacity right upper lobe present.
April 11, 1997	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
April 12, 1997	DX 16	Dr. Olson	(Negative for pneumoconiosis).[22] Advanced COPD present. (No large opacities reported).

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assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and examination, as proficient in interpreting x-ray films of all kinds including images of the lungs. *See also* 20 C.F.R. § 718.202 (a) (1) (ii). I also take judicial notice that Dr. Bassali was a B reader at the time of this interpretation. *See* B-Reader list in the Black Lung Reference section at www.oalj.dol.gov.

[20]The profusion (quantity) of the opacities (opaque spots) throughout the lungs is measured by four categories: 0 = small opacities are absent or so few they do not reach a category 1; 1 = small opacities definitely present but few in number; 2 = small opacities numerous but normal lung markings are still visible; and, 3 = small opacities very numerous and normal lung markings are usually partly or totally obscured. An interpretation of category 1, 2, or 3 means there are opacities in the lung which may be used as evidence of pneumoconiosis. If the interpretation is 0, then the assessment is not evidence of pneumoconiosis. A physician will usually list the interpretation with two digits. The first digit is the final assessment; the second digit represents the category that the doctor also seriously considered. For example, a reading of 1/2 means the doctor's final determination is category 1 opacities but he considered placing the interpretation in category 2. Additionally, according to 20 C.F.R. § 718.102 (b), a profusion reading of 0/1 does not constitute evidence of pneumoconiosis.

[21]There are two general categories of small opacities defined by their shape: rounded and irregular. Within those categories the opacities are further defined by size. The round opacities are: type p (less than 1.5 millimeter (mm) in diameter), type q (1.5 to 3.0 mm), and type r (3.0 to 10.0 mm). The irregular opacities are: type s (less than 1.5 mm), type t (1.5 to 3.0 mm) and type u (3.0 to 10.0 mm). JOHN CRAFTON & ANDREW DOUGLAS, RESPIRATORY DISEASES 581 (3d ed. 1981).

[22]Dr. Rasmussen questioned the probative value of the more recent chest x-rays in part on the basis that they were obtained during Mr. Howerton's hospitalization and he physicians were not specifically looking for pneumoconiosis. Dr. Branscomb disagreed. Since Mr. Howerton was struggling with pulmonary emboli, Dr. Branscomb would expect the hospital radiologists to have closely evaluated the chest x-rays for small lesions and reported any findings

(same)	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
April 17, 1997	DX 16	Dr. Rao	Severe chronic lung disease and fibrosis; "opacification of RUL (right upper lobe) is seen with a large cavity in its lateral- anterior aspect."
(same)	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
April 24, 1997	DX 16	Dr. Shahan	Chronic, severe irregular interstitial disease. Upper lobe, large emphysematous bulla bilaterally. (No large opacities reported).
(same)	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
April 26, 1997	DX 16	Dr. Rahman	(Negative for pneumoconiosis). Severe bilateral bullous disease; infiltrate right lower lobe. (No large opacities reported).
(same)	DX 16	Dr. Cappiello	(Negative for pneumoconiosis). Probable infiltrate right lower lobe. (No large opacity reported).
(same)	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
April 27, 1997	DX 16	Dr. Rahman	(Negative for pneumoconiosis). Severe bullous disease. (No large opacities reported).
(same)	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
April 28, 1997	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
April 30, 1997	DX 16	Dr. Cappiello	(Negative for pneumoconiosis). Infiltrate, right lower lobe. (No large opacities reported).
(same)	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
May 1, 1997	DX. 16	Dr. Rao	Severe chronic lung disease, air filed cavity right apex. (No large opacities reported).
(same)	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
May 5, 1997	DX 16	Dr. Rao	(Negative for pneumoconiosis). Severe bullous emphysema. (No large opacities reported).
(same)	DX 28	Dr. Fino, B	Negative for pneumoconiosis. Bullous emphysema present. No large opacities present.
May 11, 1997	DX 28	Dr. Wheeler, BCR, B	Negative for pneumoconiosis. Moderate COPD and emphysema present. No large opacities present.
(same)	DX 28	Dr. Scott, BCR, B	Negative for pneumoconiosis. Bullous emphysema and pneumonia present. No large opacities present.
(same)	DX 28	Dr. Scatarige, BCR, B	Negative for pneumoconiosis. Emphysema and non-specific fibrosis present. No large opacities present.

Of the 19 chest x-rays, only three films lead to interpretations of a Category A opacity. In the August 27, 1986 chest x-ray, Dr. Bassali, a dual qualified radiologist at the time, noted the presence of a size A large pulmonary opacity. However, Dr. Sargent, also a dual qualified

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of pneumoconiosis (EX 4, pages 36 and 37). I find Dr. Branscomb's position more tenable. *See Marra v. Consolidation Coal Co.* 7 BLR 1-216, 1-219 (1985) (since a physician evaluating a chest x-ray can be expected to accurately report the presence of any abnormalities, an administrative law judge may infer that the absence of a mention of pneumoconiosis indicates pneumoconiosis was not present).

radiologist, did not see a large pulmonary opacity. Due to this professional standoff, I find the August 27, 1986 chest x-ray is inconclusive for the presence of a Category A pulmonary opacity.

In the April 3, 1997 and April 6, 1997 chest x-rays, Dr. Aycoth saw a 5 cm opacity in the right upper lobe which equates to a size A pulmonary opacity.[23] However, Dr. Aycoth's large opacity finding is overwhelmed by the interpretations of the other 10 radiographic films obtained in April and May 1997. No large, size A, pulmonary opacity was reported by the physicians who reviewed the following chest x-rays: April 4, 1997, April 12, 1997, April 17, 1997,[24] April 24, 1997, April 26, 1997, April 27, 1997, April 30, 1997, May 1, 1997, May 5, 1997, and May 11, 1997.

I also note that Dr. Aycoth located the 5 cm opacity in the right upper lobe. As summarized later, when a CT scan of the lung was taken less than a month later on May 1, 1997, the radiologist did not diagnose complicated pneumoconiosis. Instead, the physician identified a 10 x 7 cm cavity in the right upper lobe associated with bullous emphysema.[25]

Accordingly, I find the preponderance of the radiographic evidence does not establish the presence of a large Category A pulmonary opacity. Further, since the preponderance of the radiographic evidence does not establish the presence of a Category A pulmonary opacity, and no biopsy or other medical tests has identified a large pulmonary mass, the record is insufficient to establish that Mr. Howerton had complicated pneumoconiosis. As a result, Mrs. Howerton may not establish the presence of pneumoconiosis through the regulatory presumption of complicated pneumoconiosis.

Chest X-Rays

The radiographic record in Mr. Howerton's case falls into two distinct time periods. First, five chest x-rays were accomplished between 1978 and 1986 for the specific purposes of assessing whether pneumoconiosis was present in Mr. Howerton's lungs. All the radiologists and physicians who evaluated the five chest x-rays from April 11, 1978, August 11, 1979, June 11, 1980, December 16, 1981, and August 27, 1986 concluded Mr. Howerton had pneumoconiosis.

However a second group of radiographic films were developed during Mr. Howerton's hospitalization in April and May 1997 for respiratory failure. In these fourteen chest x-rays, though a few physicians noted the presence of interstitial fibrosis, not one physician specifically identified pneumoconiosis. Instead, most of the doctors found severe or advanced COPD or

[23]Dr. Ahmed also observed a dense opacity in the right upper lobe; however, he did not provide any size measurement.

[24]Dr. Rao reported a "large" cavity in the right upper lobe, but did not provide any size measurement.

[25]Though Dr. Rasmussen suggested the cavity might be related to complicated pneumoconiosis (CX 1), his opinion on this issue has diminished probative value since he raised the connection as only a possibility and did not definitely diagnose complicated pneumoconiosis.

bullous disease. Additionally, several physicians concluded many of the more recent radiographic studies were specifically negative for pneumoconiosis.

Dr. Rasmussen suggested that the stark contrast between these two time periods occurred because the advanced COPD identified in the later films obscured underlying pneumoconiosis. However, Dr. Branscomb indicated that chest x-ray findings of pulmonary changes that come and go, are indicative of a disease process other than pneumoconiosis, which "evolves by getting worse."[26] Similarly, since pneumoconiosis does not go away, Dr. Fino also noted the absence of opacities in the more recent radiographic films meant the earlier images were due to some other pulmonary condition. Upon consideration of the radiographic evidence in which 14 out of the 19 chest x-rays were not positive of pneumoconiosis, I find preponderance of the radiographic studies does not establish the presence of pneumoconiosis.[27]

Medical Opinion

Although Mrs. Howerton cannot establish the presence of black lung disease in her husband's lungs through regulatory presumption or chest x-ray evidence, she may still prove this requisite element of entitlement under 20 C.F.R. § 718.202 (a) (4) through the preponderance of the more probative medical opinion. Prior to considering the various medical assessments of Mr. Howerton's pulmonary condition, a review of the other medical evidence in the record helps to understand the diverse medical opinions.

[26]September 19, 2002 deposition, page 18 (DX 28).

[27]Dr. Ramussen, Dr. Cohen, Dr. Fino, and Dr. Wheeler noted that many of the more recent x-rays from 1997 were accomplished on portable machines during Mr. Howerton's hospitalization. These physicians shared a common concern that such imagery would be of diminished quality. Dr. Cohen also indicated portable x-rays were not subject to ILO classification and Dr. Wheeler raised the possibility that some of the images may be digital. In regards to film quality, physicians addressed that concern by rating chest x-rays' film quality. Films even rated as 3 in film quality apparently remained sufficient for physicians to provide an interpretation. Concerning the purported ILO classification prohibition, many of physicians did not hesitate to render their analysis on ILO classification forms and the hospital physicians provided their findings in hospital notes, which is sufficient for my purposes. *See Consolidation Coal Co. v. Chubb,* 741 F.2d 968 (7th Cir. 1984). Finally, despite his speculation, Dr. Wheeler treated the 1997 chest x-rays as film studies rather than digital images.

Pulmonary Function Tests

Exhibit	Date / Doctor	Age / Height	FEV¹ pre[28] post[29]	FVC pre post	MVV pre post	% FEV¹ / FVC pre post	Qualified[30] pre Post	Comments
DX 1 & DX 13	June 3, 1980 Dr. Rasmussen	45 69″	2.0	3.11	88	64%	Yes[31]	Moderate obstruction
DX 2 & DX 14	Dec. 30, 1982 Dr. Rasmussen	47 69″	1.92	3.13	77	61%	Yes[32]	
DX 2 & DX 15	Aug. 25, 1986 Dr. Rasmussen	51 69”	1.41 1.76	2.88 3.36	58 79	49% 52%	Yes[33] Yes	

Arterial Blood Gas Studies

Exhibit	Date / Doctor	pCO^2 (rest) pCO^2 (exercise)	pO^2 (rest) pO^2 (exercise)	Qualified[34]	Comments
DX 2 & DX 14	Dec. 30, 1982 Dr. Rasmussen	37 37	56 57	Yes[35] Yes	
DX 2 & DX 15	Aug. 25, 1986 Dr. Rasmussen	40 44	68 48	Yes[36] Yes	
DX 16	Apr. 3, 1997 Dr. Rana	> 96.7	51	Yes[37]	(Hospitalization)
DX 16	(same) Dr. Rana	> 96.7	191	Yes	(Hospitalization)

[28]Test result before administration of a bronchodilator.

[29]Test result following administration of a bronchodilator.

[30]Under 20 C.F.R. § 718.204 (b) (2) (i), to qualify for total disability based on pulmonary function tests, for a miner's age and height, the FEV1 must be equal to or less than the value in Appendix B, Table B1 of 20 C.F.R. § 718, **and either** the FVC has to be equal or less than the value in Table B3, or the MVV has to be equal **or** less than the value in Table B5, or the ratio FEV1/FVC has to be equal to or less than 55%.

[31] The qualifying FEV1 number is 2.21 for age 45 and 69″; the corresponding qualifying FVC and MVV values are 2.77 and 88, respectively.

[32]The qualifying FEV1 number is 2.18 for age 47 and 69″; the corresponding qualifying FVC and MVV values are 2.73 and 87, respectively.

[33]The qualifying FEV1 number is 2.11 for age 51 and 69″; the corresponding qualifying FVC and MVV values are 2.66 and 84, respectively.

[34]To qualify for Federal Black Lung Disability benefits at a coal miner's given pCO^2 level, the value of the coal miner's pO^2 must be equal to or less than corresponding pO^2 value listed in the Blood Gas Tables in Appendix C for 20 C.F.R. § 718.

[35]For the pCO^2 of 37, the qualifying pO^2 is 63, or less.

[36]For a pCO^2 of 40 to 49, the qualifying pO^2 is 60, or less.

[37]For a pCO^2 above50, any pO^2 is qualifying.

DX 16	(same) Dr. Rana	54	> 250	Yes	(Hospitalization)
DX 16	Apr. 5, 1997 Dr. Rana	46	50	Yes	(Hospitalization)
DX 16	(same) Dr. Rana	54	66	Yes	(Hospitalization)
DX 16	Apr. 6, 1997 Dr. Rana	49	83	No	(Hospitalization)
DX 16	Apr. 12, 1997 Dr. Rana	86	22	Yes	(Hospitalization)
DX 16	Apr. 24, 1997 Dr. Rana	81	71	Yes	(Hospitalization)

CT Scan[38]
(DX 16)

On May 1, 1997, Dr. A.K. Rao evaluated a CT scan of Mr. Howerton's chest. The physician observed severe chronic obstructive lung disease "with extensive bullous changes" and a 10 x 7 cm cavity in the right upper lobe. Dr. Rao diagnosed severe bullous emphysema associated with a cavity in the right upper lobe.

West Virginia Occupational Pneumoconiosis Board
(DX 1 and DX 2)

On April 11, 1978, Mr. Howerton was evaluated by a team of physicians, including Dr. Walker, associated with the West Virginia Occupational Pneumoconiosis Board. Mr. Howerton was a current coal miner who complained about shortness of breath and chronic cough. Upon physical examination, suppressed breath sounds were heard. The chest x-ray showed moderately extensive pulmonary fibrosis consistent with pneumoconiosis. The physicians concluded Mr. Howerton had advanced stage occupational pneumoconiosis with a 25% impairment of pulmonary function.

On December 17, 1981, the West Virginia Occupational Pneumoconiosis Board again evaluated Mr. Howerton's pulmonary condition. Mr. Howerton continued to struggle with shortness of breath and was experiencing right chest pain. Upon physical examination, his breath sounds were normal. The chest x-ray was positive for occupational pneumoconiosis. The West Virginia Occupational Pneumoconiosis Board awarded another 15% disability due to occupational pneumoconiosis.

Mine Safety and Health Administration
(DX 2 and DX 12)

On December 10, 1985, after a medical examination determined Mr. Howerton had black lung disease, the Mine Safety and Health Administration, DOL, informed the Employer that he was eligible to exercise his option to work in a low dust area of the mine.

[38]Although several physicians referenced a second CT scan from this period, the record before me contains only this one study.

Dr. Donald L. Rasmussen
(DX 2, DX 13, DX 14, DX 15, DX 46, CX 1 and CX 4)

On December 30, 1982, Dr. Rasmussen, board certified in internal and forensic medicine, evaluated Mr. Howerton's pulmonary condition. Mr. Howerton had 27 years of coal mine employment. He had also smoked cigarettes for 20 years at the rate of half a pack a day. Upon physical examination, the breath sounds were markedly diminished. The pulmonary function test showed a moderate obstruction. The resting arterial blood gas study indicated a moderate impairment; the exercise blood gas transfer was markedly impaired. Dr. Rasmussen concluded Mr. Howerton suffered a very severe loss of respiratory functional capacity.

On August 25, 1986, Dr. Rasmussen again examined Mr. Howerton. At this time, Mr. Howerton had 29 years of coal mine employment. He also reported smoking a pack of cigarettes a day for 30 years and 1/3 a pack a day for the past 5 years. Mr. Howerton struggled with shortness of breath with exertion. His breath sounds were markedly reduced. The pulmonary function test indicated a severe obstruction that improved with bronchodilators. The arterial blood gas study revealed marked hypoxia. Dr. Rasmussen concluded Mr. Howerton was totally disabled due a respiratory disease related to his coal mine employment.

On June 24, 2004, Dr. Rasmussen reviewed Mr. Howerton's medical record and the assessments by Dr. Fino, Dr. Branscomb, and Dr. Renn. Dr. Rasmussen observed that Mr. Howerton suffered progressive impairment of his pulmonary function and struggled with pulmonary emboli. According to Dr. Rasmussen, Mr. Howerton, "die because of respiratory failure probably as a result of pulmonary embolization superimposed on his severe disabling chronic lung disease." Contrary to the impression of other medical reviewers that the radiographic evidence was negative for pneumoconiosis, Dr. Rasmussen noted that the more probative interpretations of the 1980s chest x-rays were positive for pneumoconiosis. The later chest x-rays were accomplished during hospitalization. Those poor quality studies were not analyzed for pneumoconiosis. Additionally, "x-ray appearance of simple pneumoconiosis frequently diminishes over the years as emphysema progresses." Mr. Howerton's 25 to 50 pack year history of cigarette smoking can cause chronic obstructive lung disease and emphysema. Similarly, numerous medical studies demonstrate that "coal mine dust exposure is a potent cause of chronic obstructive lung disease." According to other studies, coal miners die excessively from chronic obstructive lung disease and coal mine dust can lead to emphysema. Though other physicians have indicated that bullous emphysema is not associated with coal workers' pneumoconiosis, absent complicated coal workers' pneumoconiosis, Dr. Rasmussen believed coal mine dust exposure can cause emphysema. Specifically, "there is no pathologic reason why emphysema caused by coal mine dust should differ in its relationship to bullous emphysema than related to cigarette smoke." Both cigarette smoke and coal dust share the same cellular and biomechanics in causing lung tissue damage. Based on Mr. Howerton's pulmonary tests from the 1980s, which showed his oxygen transfer impairment exceeded his ventilatory defect, Dr. Rasmussen concluded coal dust was a significant factor in Mr. Howerton's pulmonary impairment. In conclusion, Dr. Rasmussen opined that Mr. Howerton suffered from coal workers' pneumoconiosis and related chronic obstructive lung disease. Mr. Howerton's chronic obstructive lung disease "was a major contributing factor, not only to his disability, but also to his death."

In April 2005, upon review of Mr. Howerton's 1997 hospitalization record and Dr. Ranavaya's opinion on cause of death, Dr. Rasmussen did not alter his former conclusions. He noted that the hospitalization x-ray reports indicated the presence of perihilar fibrosis and interstitial disease, which was supportive of his conclusion that Mr. Howerton had coal workers' pneumoconiosis.

Hospitalization Records and Dr. Shahib Rana
(DX 11 and DX 16)

Between April 3 and May 6, 1997, Dr. Rana hospitalized Mr. Howerton for acute respiratory failure. After worsening shortness of breath over ten days, Mr. Howerton presented at the emergency room on April 3, 1997 in severe respiratory distress with labored breathing. The physician started mechanical ventilation and placed Mr. Howerton in the intensive care unit. Mr. Howerton's past medical history included severe COPD and complicated pneumoconiosis. He had been a two pack a day cigarette smoker. Over the course of his hospitalization, Mr. Howerton's pulmonary condition vacillated several times, with at least one instance of apparent acute pulmonary failure. He was treated for pneumonia and had several chest tubes were surgically inserted to relieve pulmonary chest wall emphysema. Since Mr. Howerton had been bed-ridden for a long time and in light of his leg edema, the physicians considered the possibility of pulmonary embolus. A CT scan disclosed severe bullous emphysema. Dr. Rana diagnosed: recurrent pneumothorax, subcutaneous emphysema, right upper lobe infiltrate of an unknown etiology, severe COPD with bullous emphysema, and pneumonia.

On May 27, 1999, Dr. Rana signed Mr. Howerton's death certificate, indicating Mr. Howerton passed away on May 27,1999. The immediate cause of death was respiratory failure due to advanced COPD (chronic obstructive pulmonary disease), with pulmonary embolism a significant contributory condition. No autopsy was performed.

Dr. M. I. Ranavaya
(DX 16, CX 3, and CX 6)

On November 18, 1999, Dr. Ranavaya, board certified in occupational medicine, reviewed Mr. Howerton's medical and hospitalization record. In his opinion, Mr. Howerton had coal workers' pneumoconiosis. Although Mr. Howerton's death was not due to pneumoconiosis or complications of pneumoconiosis, Dr. Ranavaya believed pneumoconiosis was nevertheless a substantially contributing cause of his death. Mr. Howerton did not have complicated pneumoconiosis.

Dr. Ben V. Branscomb
(DX 28 and EX 4)

On July 5, July 12, and September 18, 2002, Dr. Branscomb, board certified in internal medicine, reviewed Mr. Howerton's medical and radiographic record and multiple pulmonary evaluations from 1978 through May 1999. Mr. Howerton had between 27 to 31 years of coal mine employment and smoked cigarettes as much as two packs per day for 35 years. First, Dr. Branscomb noted that while the chest x-ray interpretations had varied, the preponderance of the

radiographic evidence and the CT scan evaluation was negative for the presence of pneumoconiosis. Instead, Mr. Howerton had severe and progressive COPD with bullous emphysema, which caused recurrent respiratory infection. Additionally, the obstructive disease, coupled with pulmonary emboli, lead to necrosis in the lung zones. Such a pulmonary condition may lead to intra-pulmonary fibrosis that may resemble pneumoconiosis in a chest x-ray. Mr. Howerton died due to complication of his severe chronically disabling obstructive pulmonary disease. While coal dust exposure can produce an obstructive pulmonary defect, Mr. Howerton's "clinical course" was inconsistent with a coal dust-related pulmonary disease. As a result, even if simple pneumoconiosis was present, the changes in Mr. Howerton's pulmonary health, and the mode and timing of his death, indicate his pulmonary obstruction was not due to coal mine dust exposure. However, no objective medical evidence indicated the presence of either medical or legal pneumoconiosis. Mr. Howerton was totally disabled due to bullous emphysema.

In a September 19, 2002 deposition, Dr. Branscomb reiterated that Mr. Howerton did not have either simple or complicated pneumoconiosis. Significantly, none of the more recent chest x-rays showed any lesions compatible with complicated pneumoconiosis. At one time, Mr. Howerton did have a pulmonary abscess which some physicians may have incorrectly believed resembled complicated pneumoconiosis. Though Dr. Rasmussen's earlier diagnosis of pleurisy, or inflammation of the pleural is not associated with pneumoconiosis, that condition helps explain Mr. Howerton's multiple hospitalizations for lung infections and pneumonia. Additionally, individuals with severe emphysema often suffer recurrent lung infections. Mr. Howerton's clinical presentation was typical for a heavy cigarette smoker. He had a significant history of cigarette smoking and continued to smoke cigarettes even after recurring exacerbation of his obstructive lung disease with infection.

In a June 1, 2005 deposition, Dr. Branscomb reconsidered Mr. Howerton's pulmonary condition, having reviewed the recent assessments by Dr. Fino, Dr. Rasmussen, and Dr. Cohen. Dr. Branscomb agreed that coal dust inhalation can produce an obstructive impairment. However, in his opinion, "there are no cases anywhere in the records of anyone ever developing a bullous emphysema with a pneumothorax and a [chest] tube put in attributable to coal mine dust." Only in one situation, when complicated coal workers' pneumoconiosis produces a large contracted portion of the lung, can a type of bullous emphysema, called traction emphysema, be produced. However, though he suffered with bullous emphysema, Mr. Howerton did not also have complicated coal workers' pneumoconiosis. The earlier lesions that may have suggested the presence of complicated pneumoconiosis later resolved and were probably pulmonary emboli. Further, while bullous emphysema is a regular occurrence with cigarette smoking, it is not associated with coal mine dust inhalation. Mr. Howerton's cigarette smoking related COPD contributed to his death. Additionally, "pulmonary emboli was a very significant and major component" because Mr. Howerton's death was sudden and he had a significant problem with emboli such that a filter had been inserted in his leg vein in an attempt to block emboli from traveling to his lungs. Emboli had also caused some lung necrosis and the development of a cavity in his lungs with a corresponding infection. Severe emphysema and pulmonary emboli are "closely related."

Dr. Joseph J. Renn, III
(DX 28 and EX 1)

On July 11, 2002, Dr. Renn, board certified in pulmonary disease and internal medicine, also reviewed Mr. Howerton's extensive medical record. Mr. Howerton had worked in coal mines for over thirty years. His reported cigarette smoking history varied from 20 to 70 pack years.[39] Beginning in the early 1990s, he received medical treatment and was hospitalized for leg edema, pulmonary distress, and pneumonia. The preponderance of the chest x-ray interpretations were negative for pneumoconiosis. Similarly, most interpretations of a May 1997 CT scan were negative for pneumoconiosis and instead demonstrated the presence of COPD. A June 1997 CT scan also indicated the presence of COPD. Based on his review of the medical history and objective medical evidence, Dr. Renn opined Mr. Howerton most likely died due to recurrent pulmonary emboli. His centrilobular, panlobular, and bullous emphysema was due to tobacco smoking. He did not have coal workers' pneumoconiosis. During the later portion of his life, Mr. Howerton was permanently and totally disabled due to tobacco smoke-induced emphysema and chronic bronchitis with an asthmatic component. His death was neither caused nor contributed to by pneumoconiosis.

On April 20, 2005, after reviewing Dr. Rasmussen's more recent assessments, Dr. Renn provided additional comments about Mr. Howerton's death and the relationship between coal dust exposure and lung disease. Noting the Mr. Howerton died "suddenly" and was declared "deceased on arrival" at the hospital, Dr. Renn indicated his death was "most probably a result of a pulmonary emboli." Dr. Renn acknowledges that coal mine dust can cause an obstructive airways disease. Initially, the lung tissue reacts the same to the "insult" caused by either coal dust or tobacco smoke. However, the subsequent "mechanisms of destruction is vastly different." Coal dust produces fibrosis and focal emphysema in the primary and secondary bronchiole areas rather than the toxin inflammation produced by cigarette smoke. Though coal dust and cigarette smoke can cause emphysema, "most types of emphysema are caused by tobacco smoking." Whereas, "only focal and . . . centrilobular emphysema are types known to be caused by coal mine dust exposure." At the same time, bullous emphysema does not occur in association with simple coal workers' pneumoconiosis. Dr. Renn again noted that the preponderance of chest x-ray evidence was negative for pneumoconiosis. Though a few dual qualified radiologist observed pneumoconiosis in a 1986 film, they determined pneumoconiosis was not present in the more recent chest x-rays. Additionally, a CT scan, which is a more sensitive detection device, was negative for pneumoconiosis and positive for bullous emphysema. Bronchodilator-reversibility is inconsistent with coal workers' pneumoconiosis; instead, it is a "pattern associated with tobacco smoking." Similarly, considering the abnormal diffusion test results, Mr. Howerton's gas exchange impairment being greater than his pulmonary ventilatory problem is not unexpected. The objective medical tests support a conclusion that Mr. Howerton's COPD and bullous emphysema are due to his years of tobacco smoking.

[39]A pack year equals the consumption of a pack of cigarettes a day for one year.

Dr. Gregory J. Fino
(DX 28, EX 2, and EX 3)

On July 16, 2002, Dr. Fino, board certified in pulmonary disease and internal medicine, reviewed Mr. Howerton's medical record. Mr. Howerton mined coal for 30 years and had a 70 pack year history of cigarette smoking. In assessing the cause of Mr. Howerton's severe pulmonary impairment, Dr. Fino first noted the variability over the course of years in the arterial blood gas studies. Such variability was inconsistent with progressive pneumoconiosis and consistent with severe bullous emphysema, collapsed lung, and pneumonia. Second, Dr. Fino observed that in addition to the negative chest x-rays, the CT scan was "especially striking" for very severe bullous emphysema, which is unrelated to coal mine dust exposure and simple pneumoconiosis. Based on the great variability in arterial blood gas studies, radiographic evidence, and the CT scan, Dr. Fino opined that very severe bullous emphysema due to cigarette smoking caused Mr. Howerton's respiratory impairment, pulmonary disability, and death. The development of blood clots to the lungs was a significant contributing factor. Mr. Howerton's exposure to coal mine dust did not cause, contribute to, or hasten his death. He did not have pneumoconiosis or complicated pneumoconiosis.

In a September 19, 2002 deposition, Dr. Fino stated that Mr. Howerton did not have simple or complicated pneumoconiosis. In his review of numerous chest x-rays, Dr. Fino did not observe pneumoconiosis or an opacity greater than one centimeter. Dr. Fino also noted that the smaller opacities identified in the 1980s were no longer present in the films he reviewed from the 1990s. Since pneumoconiosis does not go away, those earlier opacities were due to some other pulmonary condition. Similarly, a CT scan was negative for complicated pneumoconiosis. A CT scan is very helpful in diagnosing a pulmonary condition because it's more sensitive than chest x-rays. Based on the objective medical evidence, Dr. Fino concluded Mr. Howerton had severe bullous emphysema which is "large, dilated destroyed air sacs in his lungs." The condition is unrelated to coal dust exposure. The pulmonary function test response to bronchodilator treatment points to a cigarette smoking-related pulmonary disease because a coal dust-related pulmonary condition does not respond to bronchodilators. The variable hypoxia demonstrated over the course of years in the arterial blood gas studies is also inconsistent with pneumoconiosis. Because he was predisposed due to cigarette smoking-related bullous emphysema, Mr. Howerton had a significant problem with lung infections. Further, due to his pulmonary impairment, Mr. Howerton did not move around and thus developed blood clots in the veins of his legs which "traveled to his lungs producing a blood clot in the lungs." Mr. Howerton died a pulmonary death due in part to the pulmonary blood clots.

In April 2005, Dr. Fino reviewed Dr. Rasmussen's recent assessments and further evaluated Mr. Howerton's pulmonary condition. Dr. Fino continued to believe that Mr. Howerton had been totally disabled due to very serious bullous emphysema, attributable to cigarette smoking. He believed the effects of cigarette smoke and coal mine dust on the lungs could be distinguished. While simple coal worker's pneumoconiosis may cause focal emphysema, it is not associated with bullous emphysema. Based on his review and critiques of multiple medical studies, Dr. Fino concluded to opine that Mr. Howerton's pulmonary disability and death related to his inhalation of cigarette smoke.

In an April 28, 2005 deposition, Dr. Fino again reviewed Mr. Howerton's case, having considered Dr. Cohen's review. Mr. Howerton had a severe pulmonary impairment and disability due to cigarette smoking "which contributed to his death and he also had blood clots to his lungs at the time of death which was probably the final straw for this gentlemen." Mr. Howerton had two pulmonary risk factors, 29 years of coal mine employment and 20 to 70 pack years of cigarette smoking. In determining the effect of those two risks, Dr. Fino was most influenced by the CT scan showing "very severe bullous emphysema." The finding was significant because bullous emphysema is not a type of emphysema associated with the inhalation of coal dust inhalation, absent the presence of complicated pneumoconiosis, which Mr. Howerton did not have. According to Dr. Fino, medical studies indicate that "bullous emphysema is very classic for a non-coal dust related condition." Also importantly, the CT scan did not show "any evidence of coal dust retention in the lung." The negative radiographic evidence was also significant because when pneumoconiosis causes an obstructive pulmonary defect, its presence in the lungs is substantial. Mr. Howerton's radiological record did not show the presence of substantial pneumoconiosis. Dr. Fino also emphasized that the variability in arterial blood gas studies is diagnostic. Such variability suggests another pulmonary condition because pneumoconiosis is a fixed, permanent pulmonary condition. In Mr. Howerton's case, "the other condition would be a smoking-related abnormality." For these reasons, Dr. Fino concluded Mr. Howerton's pulmonary impairment and death were due to cigarette smoking.

Dr. Robert A.C. Cohen
(CX 2 and CX 5)

On April 14, 2005, Dr. Cohen, board certified in pulmonary disease, internal medicine, and critical care, reviewed Mr. Howerton's medical record, numerous pulmonary evaluations, radiographic findings and the assessments of Dr. Rasmussen, Dr. Branscomb, Dr. Fino, and. Dr. Renn. Mr. Howerton had been a coal miner for at least 24 years. His cigarette smoking cigarette smoking history varied from 15 to 60 pack years. Based on "significant" radiographic evidence, coupled with clinical and physiological presentations, Dr. Cohen opined Mr. Howerton had coal workers' pneumoconiosis. Similarly, Dr. Cohen attributed Mr. Howerton's chronic, severe obstructive defect and diffusion impairment to both his 24 year exposure to coal mine dust and 15 to 60 pack year history of cigarette smoking. Concerning the connection between coal mine dust exposure and obstructive pulmonary disease, Dr. Cohen emphasized several points. First, coal mine dust can cause an obstructive impairment. Notably, cigarette smoking is not the sole cause of emphysema. Coal mine dust can cause all types of emphysema, including bullous emphysema. Second, exposure to one pulmonary risk factor, such as cigarette smoke, does not insulate a person from the harmful effects of a second pulmonary risk factor, such as coal mine dust. Third, variability in arterial blood gas studies can be attributed to exacerbation of a pulmonary condition due to infection and thus is not indicative of etiology. Significantly, the "severe gas exchange abnormalities exceeded his ventilatory impairment, which is indicative of a disease process induced by coal mine dust." Fourth, no medical or scientific basis exists for concluding that response to bronchodilator permits distinguishing COPD due to cigarettes from COPD due to coal mine dust. Fifth, medical studies support a connection between coal mine dust exposure and an obstructive pulmonary defect "not far less frequent than that from exposure to cigarette smoking." Sixth, medical studies provide a firm foundation to reasonably conclude with a long term cigarette smoker and coal miner, such as Mr. Howerton, "both factors

contributed to, or caused, the impairment." In light of these considerations, Dr. Cohen determined that Mr. Howerton was totally disabled due his pulmonary impairment caused by both his exposure to coal mine due and cigarette smoking. In Dr. Cohen's opinion, Mr. Howerton "clearly died as a result of his pulmonary disease" caused by his coal mine employment and cigarette smoking.

Discussion

The physicians with the West Virginia Occupational Pneumoconiosis Board, the Mine Safety and Health Administration, Dr. Ranavaya, Dr. Rasmussen and Dr. Cohen concluded that Mr. Howerton had pneumoconiosis. Dr. Branscomb, Dr. Renn, and Dr. Fino reached a different conclusion and found that Mr. Howerton did not have pneumoconiosis. Finally, Dr. Rana neither diagnosed pneumoconiosis nor identified the cause of Mr. Howerton's severe COPD and associated bullous emphysema. Due to this conflict in medical opinion, I must first assess the relative probative value of each respective opinion in terms of documentation and reasoning.

Regarding the first probative value consideration, documentation, a physician's medical opinion is likely to be more comprehensive and probative if it is based on extensive objective medical documentation such as radiographic tests and physical examinations. *Hoffman v. B & G Construction Co.*, 8 B.L.R. 1-65 (1985). In other words, a doctor who considers an array of medical documentation that is both long (involving comprehensive testing) and deep (includes both the most recent medical information and past medical tests) is in a better position to present a more probative assessment than the physician who bases a diagnosis on a test or two and one encounter.

The second factor affecting relative probative value, reasoning, involves an evaluation of the connections a physician makes based on the documentation before him or her. A doctor's reasoning that is both supported by objective medical tests and consistent with all the documentation in the record, is entitled to greater probative weight. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). Additionally, to be considered well reasoned, the physician's conclusion must be stated without equivocation or vagueness. *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91 (1988).

With these principles in mind, I first find Dr. Ranavaya's terse comments and responses concerning Mr. Howerton's pulmonary condition have little probative value due to the absence of any reasoning. Having reviewed the medical record, Dr. Ranavaya had a good documentary basis for his determinations. However, without presenting any rationale or explanation, Dr. Ranavaya simply indicated that Mr. Howerton had coal workers' pneumoconiosis.

Next, the finding of coal workers' pneumoconiosis by the West Virginia Occupational Board has diminished probative value due incomplete documentation and the absence of sufficient reasoning. The panel of physicians essentially relied on positive chest x-rays from 1978 and 1981 for their diagnosis. However, due to the dated nature of their assessments, the physicians were not aware of the substantial number of subsequent negative chest x-rays obtained in 1997 and that the preponderance of the radiographic evidence is actually negative for the presence of coal workers' pneumoconiosis. To the extent the physicians also relied on the

physical examination results for the pneumoconiosis diagnosis, their finding is diminished due to the absence of any explanation about how the results of the examination supported a finding of legal pneumoconiosis.

For similar reasons, incomplete documentation and insufficient reasoning, the conclusion by the Mine Safety and Health Administration has diminished probative value. Relying on one medical examination in 1985, the administration officials were unaware that the preponderance of the radiographic evidence was negative for pneumoconiosis. More significantly, the officials only provided conclusions and did not address or even identify the aspects of the examination that supported their finding Mr. Howerton had black lung disease.

Turning to Dr. Rana, based on his April/May 1997 hospitalization of Mr. Howerton and the numerous tests and radiographic studies, the physician had a firm foundation for his diagnoses. In light of that documentation, the absence of a diagnosis of clinical pneumoconiosis has some probative value. At the same time, Dr. Rana's silence on etiology of the COPD does not help in ascertaining whether Mr. Howerton had legal pneumoconiosis.

Having conducted two pulmonary examinations of Mr. Howerton and reviewed the medical record, Dr. Rasmussen had a generally solid foundation for his conclusions about Mr. Howerton's pulmonary conditions. Nevertheless, Dr. Rasmussen's finding of clinical coal worker's pneumoconiosis has lessened probative value due to inaccurate documentation. Believing the 1997 chest x-rays had little probative value and thus based solely on the films from the 1970s and 1980s, Dr. Rasmussen opined that the radiographic record showed the presence of coal workers' pneumoconiosis. To the contrary, I have determined that more recent chest x-rays have probative value and the preponderance of the radiographic evidence is actually negative for the presence of coal workers' pneumoconiosis.

On the issue of legal pneumoconiosis, Dr. Rasmussen has presented a documented, reasoned, and probative assessment. For two principle reasons, Dr. Rasmussen believes Mr. Howerton's obstructive impairment is due to his exposure to coal mine dust. First, since cigarette smoke and coal mine dust produce the same chemical effects on the lungs, no pathologic reason exits to conclude that coal mine dust would not cause bullous emphysema as well as cigarette smoke. Second, as demonstrated by the arterial blood gas studies and pulmonary function test, Mr. Howerton's gas transfer exchange impairment is much greater than his loss of pulmonary function. This pattern is indicative of a pulmonary impairment attributable to coal mine dust inhalation.

Based on an extensive review of the medical record and numerous pulmonary evaluation, Dr. Branscomb provided a documented, reasoned, and probative determination that Mr. Howerton had neither clinical nor legal coal workers' pneumoconiosis. Regarding clinical pneumoconiosis, Dr. Branscomb noted that the preponderance of the radiographic evidence and a CT scan were negative for the presence of pneumoconiosis. Concerning legal pneumoconiosis, although Mr. Howerton was severely disabled by, and died in part as a result of, very severe bullous emphysema, Dr. Branscomb found no connection with that particular type of emphysema and Mr. Howerton's coal mine employment. While exposure to coal mine dust can cause emphysema, Dr. Branscomb indicated Mr. Howerton's clinical presentation of bullous

emphysema was a classic case for a significant cigarette smoker. Significantly, according to Dr. Branscomb no medical studies showed a connection between the development of bullous emphysema and exposure to coal mine dust, in the absence of complicated pneumoconiosis.

Dr. Renn also developed a reasoned and probative evaluation of Mr. Howerton's pulmonary condition based on solid documentation from an extensive review of the medical record and pulmonary evaluations. Dr. Renn concluded Mr. Howerton did not have clinical pneumoconiosis because the preponderance of the chest x-rays were negative, as well a CT scan, a more sensitive diagnostic tool. Further, Mr. Howerton did not have legal pneumoconiosis because his bullous emphysema was not related to his coal mine employment. While coal mine dust can cause a pulmonary obstruction, a distinction can be made between a cigarette smoke-related and a coal dust-related obstruction. Initially, both cigarette smoke and coal mine dust cause a similar "insult" to the lung tissue. However, the eventual destruction of the lung tissue by these two pulmonary irritants is "vastly" different. Cigarette smoke can cause most types of emphysema. Whereas, coal mine dust only causes one type of emphysema, focal, and it occurs only in the primary and secondary and bronchiole areas. Mr. Howerton did not have focal emphysema. Instead, he suffered with bullous emphysema with has been identified with cigarette smoking. Dr. Renn also note the reversibility demonstrated in the pulmonary function tests was inconsistent with pneumoconiosis and instead associated with cigarette smoking. Finally, considering the abnormal diffusion test results, the disparity in severity between the gas transfer impairment and loss of pulmonary function is not unexpected.

In a similar manner, based on an extensive review of the medical evidence, Dr. Fino reached documented, reasoned, and probative conclusions about Mr. Howerton's pulmonary ailment. Based on the preponderance of the radiographic evidence and a negative CT scan, Dr. Fino reasonably concluded Mr. Howerton did not have clinical pneumoconiosis. Addressing the presence of legal pneumoconiosis, Dr. Fino indicated that several aspects of Mr. Howerton's case pointed to cigarette smoke as the cause of his pulmonary obstruction. First, the variability in the arterial blood gas studies was inconsistent with pneumoconiosis which causes a fixed and permanent impairment. Second, for the same reason, the response of Mr. Howerton's pulmonary obstruction to bronchodilator therapy pointed to cigarette smoke as the appropriate etiology since a coal mine dust induced obstruction does not improve. Third, the etiology of Mr. Howerton's emphysema is discernible because medical studies demonstrate that pneumoconiosis causes an obstructive pulmonary impairment through focal emphysema rather than bullous emphysema which was extensively present in Mr. Howerton's lungs. Bullous emphysema is a classic form of a non-coal dust related pulmonary condition.

Dr. Cohen also conducted a thorough review and analysis of Mr. Howerton's medical record and the numerous pulmonary evaluations and assessments. Citing "substantial" radiographic evidence of pneumoconiosis, plus clinical and physiological findings, Dr. Cohen diagnosed clinical coal workers' pneumoconiosis. However, I have determined that the preponderance of the radiographic evidence is in effect substantially negative for pneumoconiosis. Consequently, based on his stated reliance of a substantial radiographic record that is contrary to my determination, Dr. Cohen's diagnosis of clinical pneumoconiosis has diminished probative value.

At the same time, in a documented, reasoned and probative opinion, Dr. Cohen has also diagnosed legal pneumoconiosis. Based on several factors, Dr. Cohen attributes Mr. Howerton's severe bullous emphysema to both his extensive cigarette smoking and long-term coal mine employment. First, medical studies have established that coal mine dust can cause all types of emphysema, including bullous emphysema. Second, the variability in arterial blood gas studies in Mr. Howerton's case is due to his recurrent pulmonary infection and does not provide any evidence on the etiology of his emphysema. Third, Mr. Howerton's arterial blood gas transfer impairment if greater than his pulmonary function deficiency. That situation is indicative of a coal mine dust related obstructive impairment. Fourth, no medical study has demonstrated that a bronchodilator response identifies coal mine dust rather than cigarette smoke as the cause of an obstructive impairment.

In summary, for documentation and reasoning shortfalls, the opinions of the Dr. Ranavaya, the West Virginia Occupational Pneumoconiosis Board, and the Mine Safety and Health Administration have diminished probative value.

Concerning the presence of clinical coal workers' pneumoconiosis, the opinions of Dr. Rasmussen and Dr. Cohen have diminished probative due their reliance on radiographic documentation that is inaccurate. In contrast, consistent the radiographic and CT scan evidence, Dr. Rana did not include clinical pneumoconiosis in his hospital discharge diagnosis and Dr. Branscomb, Dr. Renn, and Dr. Fino, in reasoned, documented, and probative assessments, opined Mr. Howerton did not have clinical coal workers' pneumoconiosis. Accordingly, the preponderance of the more probative medical opinion establishes that Mr. Howerton did not have clinical coal workers' pneumoconiosis.

In regards to the presence of legal pneumoconiosis, Dr. Rasmussen, Dr. Branscomb, Dr. Renn, Dr. Fino, and Dr. Cohen have presented well documented and reasoned medical opinions. Having reviewed the same objective medical evidence and relying on essentially the same medical studies, these highly qualified medical experts disagree on whether a connection existed between Mr. Howerton's exposure to coal mine dust and his severe bullous emphysema. Despite extensive consideration of their respective opinions on the etiology of Mr. Howerton's bullous emphysema, I am unable to sufficiently differentiate as more probative the starkly contrary views of Dr. Rasmussen, Dr. Cohen, Dr. Branscomb, Dr. Renn, and Dr. Fino. The conclusions by Dr. Branscomb, Dr. Renn, and Dr. Fino that Mr. Howerton does not have legal pneumoconiosis outweighs the other two contrary opinions of Dr. Rasmussen and Dr. Cohen and seemingly represent the preponderance of the medical opinion. Yet, due to the indistinguishable probative value of the five physicians' analyses on whether coal mine dust inhalation can cause bullous emphysema, I conclude the more probative medical opinion on the presence of legal pneumoconiosis effectively stands in an evidentiary equipoise. Consequently, due to that medical expert impasse, Mrs. Howerton is unable to establish that her husband had legal coal workers' pneumoconiosis through the preponderance of probative medical opinion under 20 C.F.R. § 718.202 (a)(4).

Compton Analysis

Under the guidance of the decision in *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000), I must also consider both the chest x-ray evidence and medical opinion together to determine whether Mr. Howerton had pneumoconiosis. In that regard, since standing alone neither the preponderance of the chest x-rays nor probative medical opinion establish the presence of pneumoconiosis, consideration of that evidence together obviously still fails to produce a finding of pneumoconiosis.

CONCLUSION

The preponderance of the radiographic evidence and more probative medical opinion establishes that Mr. Howerton did not have either complicated or simple coal workers' pneumoconiosis. The more probative medical opinion on whether Mr. Howerton had legal pneumoconiosis based on a connection between his severe pulmonary obstructive impairment and inhalation of coal mine dust stands in equipoise. As a result, Mrs. Howerton is unable to prove the presence of legal pneumoconiosis in her husband's lungs. Accordingly, having failed to establish a requisite element of entitlement in a survivor claim, the presence of pneumoconiosis, Mrs. Howerton's modification request must be denied.

ORDER

The modification request by MRS. PEGGY J. HOWERTON is **DENIED**.

SO ORDERED:

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RICHARD T. STANSELL-GAMM
Administrative Law Judge

Date Signed: July 20, 2006
Washington, DC

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. See 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. See 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. See 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).